Correspondence

The Editorial Board will be pleased to receive and consider for publication correspondence containing information of interest to physicians or commenting on issues of the day. Letters ordinarily should not exceed 600 words, and must be typewritten, double-spaced and submitted in duplicate (the original typescript and one copy). Authors will be given an opportunity to review any substantial editing or abridgement before publication.

Sleep Apnea Studies

TO THE EDITOR: I am writing in regard to the article "Sleep Apnea: A Prospective Study," published in the August 1983 issue.[1] One of the conclusions of the article is that older patients may deserve routine assessment in screening for sleep apnea. In my opinion this falls into the category of treating a test rather than a patient. I believe that most sleep laboratories require (certainly ours does) oxygen desaturation as well as clinical symptoms to define a "syndrome of sleep apnea." In these days of efforts to try and control medical costs, routine screening of all types must be thought out very carefully and in particular a test that carries the kind of expense that routine apnea monitoring or screening would entail.

It is well recognized that both children and elderly patients have more sleep apnea. I think the data provided by monitoring a group of asymptomatic patients with sleep apnea in a research facility may be of value over the long term. This sort of data along with oxygen saturation monitoring associated with the prospective observation of these patients for the development of pathologic features such as sleep disturbance, sudden death, congestive failure or respiratory dysfunction will be of great help. I do think, however, that to make any recommendations for evaluation or therapy on the basis of this study would be a mistake.

W. DALE OVERFIELD, DDS, MD
Tacoma, Washington

REFERENCE

1. Kreis P, Kripke DF, Ancoli-Israel S: Sleep apnea: A prospective study. West J Med 1983 Aug; 139:171-173

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Drs Kreis, Kripke and Ancoli-Israel Respond

TO THE EDITOR: We certainly agree with Dr Overfield that physicians should treat the whole patient and not a single test. We also agree that portable screening recordings do not provide a complete apnea evaluation any more than a routine electrocardiogram provides a complete cardiac evaluation, and that one would not initiate a major treatment for sleep apnea such as long-term medication or head and neck surgical procedures without thorough clinical evaluation. If a clinical examination suggests that the patient may be suffering significant symptoms or significant cardiorespiratory impairment related to sleep apnea, then further testing such as all-night oximetry would be indicated.

Nevertheless, a portable screening recording would be sufficient by itself to provide clinical guidance in some areas. Hypnotic drugs and other sedatives should generally be withdrawn from patients shown to have several hundred episodes of apnea a night. In addition, such patients should be warned about the deleterious effects of drinking alcohol before sleep. Patients with obstructive apnea who are obese are likely to benefit from weight loss. If a patient with central apnea is receiving alkalinizing diuretics, addition or substitution of acetazolamide would be reasonable. Such simple and practical recommendations could be made on the basis of an inexpensive screening test.

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Chronic Arsenic Poisoning

TO THE EDITOR: The recent case report by Selzer and Ancel on chronic arsenic poisoning[1] underscores the importance of including toxicologic factors in our attempts at differential diagnosis. Unfortunately, valuable data were not included in the attempt to locate the source of the arsenic exposure in this case. The hair is particularly useful as a biopsy material in determining the time of exposure: each inch away from the root takes us back about two months in time so that sampling the hair over its entire length can yield time-related information.

I was surprised to find no follow-up testing to assure that toxic exposure had ceased. Finally, in view of the poor response to treatment, would it not be worthwhile to include testing of physiologic minerals (such as zinc, magnesium and manganese) with which heavy metals are known to interfere? Treating such mineral deficiencies can attenuate the toxic effects of heavy metals so our interest in them is quite practical from the clinical point of view.

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REFERENCE

1. Selzer PM, Ancel MA: Chronic arsenic poisoning masquerading as pernicious anemia. West J Med 1983 Aug; 139:219-220

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Dr Selzer Responds

TO THE EDITOR: I should like to make the following comments in response to Dr Kunin's letter to the editor.

A discussion detailing the search for a source of arsenic poisoning was intentionally excluded from our report in the interest of brevity and because we felt it to be tangential to the emphasis of the paper. The results of hair analysis, however, are indeed mentioned, and it was on this basis that we determined the subacute rather than chronic nature of the toxic exposure.

Law enforcement as well as occupational health and safety officials were notified and an examination of the home and work environment ensued. Family members also had their urine screened for heavy metals. While not involved in this undertaking, it was our understanding that a thorough investigation revealed no environmental toxic source.

We believed that the patient's response to chelation and his overall clinical course were typical of those described in the literature.[1] Given the results of the investigation, the patient's transfer to a nursing facility after hospital discharge and his continued gradual neurologic improvement, it was felt that the risk of reexposure was minimal. Hence, no follow-up toxicologic studies were performed. Adequate caloric and nutritional support was maintained during his recovery.

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REFERENCE

1. Heyman A, Pfeiffer JB Jr, Willet RW, et al: Peripheral neuropathy caused by arsenical intoxication: A study of 41 cases with observations on effects of BAL (2,3-dimercaptopropanol). N Engl J Med 1956; 254: 401-409

Cellular Sodium Transport and Hypertension: A New Hypothesis

TO THE EDITOR: In Tuck's informative and interesting contribution "Sodium Transport in Hypertension" to the "Hypertension Symposium: Newer Topics on Normal and Abnormal Blood Pressure Regulatory Mechanisms"[1] the question is raised as to whether sodium transport abnormalities in erythrocytes "reflect the sodium and potassium ion transport capacity of more relevant target tissues such as vascular smooth muscle in essential hypertension." For obvious ethical reasons it is not feasible to study sodium transport in excised segments of human arterial wall. There are, however, several animal models where the hypothesis of abnormal sodium transport in vascular tissues has been tested.

In spontaneously hypertensive rats we found[2] an augmentation of the extracellular to intracellular Na^+ ratio in arterial wall attributable to a relative increment of extracellular Na^+. This change is consistent with a stimulation of the sodium-potassium pump; such an increase in pump activity in spontaneously hypertensive rats has been shown by Pamnani and co-workers.[3]

On the other hand, in arterial wall of rats with saline-induced hypertension we found[4] a diminution of extracellular to intracellular Na^+ ratio, thus suggesting an increment of Na^+ into cells. This would be consistent with decreased activity of the Na^+-K^+ pump; indeed such a decrease in pump activity for the saline model of hypertension has been shown by Pamnani and associates.[3] Once more, studies in animals help provide a sound physiopathological interpretation to clinical observations, which unavoidably have to be more limited in scope.

We earlier concluded[2,4] that hypertension may be related to changes in arterial wall smooth muscle electrolyte distribution and in a recent paper we presented

> the hypothesis, as provocative as it may appear, that the change from human essential hypertension to malignant hypertension may well be associated with a change in the vascular wall from increased extracellular Na^+ to augmented intracellular Na^+ caused by suppression of the Na^+-K^+ pump mediated by a superimposed agent, conceivably of the type of the demonstrated ouabainlike humoral factor.[5]

To close, there are three points I wish to emphasize: (1) As stated by Tuck in the symposium[1] it is now clear that hypertension cannot be regarded as a relatively uniform disease having one predominant cause. (2) I very much doubt that useful diagnoses or predictions in regard to hypertension may be obtained from studying sodium transport in nonsolid human tissues such as blood cells, particularly erythrocytes that, at least in humans, are incomplete and anucleated cells. Transport of Na^+, and other ions, must be studied in the arterial wall, which ultimately is the common pathway for the manifestation of hypertension. (3) That the principal ion transport aberration in essential hypertension may be an increase in *intra*cellular Na^+ appears too drastic a change; it can be surmised from cell physiology considerations that excessive intracellular Na^+ is far more harmful to cellular function than increased extracellular Na^+. Thus, our findings[2,4] combined with those of Pamnani and co-workers[3] on the Na^+-K^+ pump provide strong support for placing the increased Na^+, at least during the long, controllable phase of hypertension, in the extracellular space..

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REFERENCES

1. Tuck ML, Golub MS, Eggena P, et al: Hypertension symposium: Newer topics on normal and abnormal blood pressure regulatory mechanisms. West J Med 1983 Aug; 139:190-203

2. Llaurado JG, Madden JA: Sodium kinetics in aorta of spontaneously hypertensive rats. J Appl Physiol 1975 Nov; 39:868-872

3. Pamnani M, Huot S, Buggy J, et al: Demonstration of a humoral